105. GASCHROMATOGRAPHIC DETERMINATION OF POLYCYCLIC AROMATIC HYDROCARBONS, AZA-ARENES, AROMATIC AMINES IN THE PARTICLE AND VAPOR PHASE OF MAINSTREAM AND SIDESTREAM SMOKE OF CIGARETTES

Grimmer G, Naujack KW, Dettbarn G
Biochemisches Institut fur Umweltcarcinogene, Grosshansdorf, F.R.G.
Toxicol Lett; 35(1):117-24 1987

The present arrangement collects particles and semivolatiles of main- and sidestream smoke and allows a recovery of the trapped substances nearly quantitatively and without impurities. The fractionation procedure allows to separate various groups of carcinogens such as PAH, aza-arenes and aromatic amines for analytical and biological studies. Sidestream smoke contains ten times more polycyclic aromatic hydrocarbons (PAH) compared with mainstream smoke. This holds also true for aza-arenes and amines. PAH of the gaseous phases include only 1% of the particle-bound PAH.

106. PILOT STUDY OF SAMPLING AND ANALYSIS FOR POLYNUCLEAR AROMATIC COMPOUNDS IN INDOOR AIR

Chuang JC, Mack GA, Koetz JR, Petersen BA
Battelle Columbus Div., OH
Available from National Technical Information Service, Springfield, VA as NTIS/PB87-129524. 223 p., 1987.

The report presents the results of a nine-home indoor air study that the authors performed in Columbus, Ohio in the winter of 1984. The main objective of the study was the field evaluation of sampling and analysis methodology. However, several additional results of the study are interesting and useful, including the finding of polynuclear aromatic hydrocarbons (PAH) at levels from 0.000 to 143 ng/m3 in residences, strong correlations of these levels with smoking by occupants, little influence of fireplace use on the PAH levels, the identification of quinoline as a potential marker for cigarette smoking, and measurement of 2- and 3-nitrofluoranthene, which are potent mutagens, at significant levels from 0.000 to 0.185 ng/m3, also correlated with smoking.

107. GENOTOXICITY AND POLYNUCLEAR AROMATIC HYDROCARBON ANALYSIS OF ENVIRONMENTAL TOBACCO SMOKE SAMPLES FROM RESTAURANTS

Husgafvel-Pursiainen K, Sorsa M, Miller M, Benestad C
Institute of Occupational Health, Helsinki, Finland.
Mutagenesis; 1(4):287-92 1986

Acetone-extracted samples of airborne particulate matter collected in three restaurants were analysed for their content of polynuclear aromatic hydrocarbons (PAH) and related polynuclear aromatic compounds (PAC) as well as for genotoxic activity using the Salmonella/microsome assay (strains TA98 and TA100) and sister chromatid exchange (SCE) induction in Chinese hamster ovary (CHO) cell cultures. The total particulate matter varied considerably in the restaurants, being 1.37 mg/m3 at the highest; in the same restaurant the highest amount of total PAHs (168 ng/m3) was also detected. Altogether, 13-22 individual PACs were identified in the samples, ranging from phenanthrene to benzothionaphthene. All of the six samples caused significant increases both in bacterial revertant and SCE frequencies. In the Salmonella assay, the mutagenic activity detected was primarily with metabolic activation. However, in the CHO cell cultures the induction of SCEs was also seen without an exogenous metabolic activation system. The cytotoxicity of the extracts limited the concentration range tested in the SCE assay. Only a partial correspondence of the total PAH content with the genotoxic activity of the samples was found. The genotoxicity of restaurant air exceeded by one to two orders of magnitude the previously reported activities detected by similar methods in urban outdoor and indoor air samples.

108. NOTE ON ETHENE AND OTHER LOW-MOLECULAR WEIGHT HYDROCARBONS IN ENVIRONMENTAL TOBACCO SMOKE

Persson KA, Berg S, Tornqvist M, Scalia-Tomba GP, Ehrenberg L
Department of Analytical Chemistry, University of Stockholm, Sweden.
Acta Chem Scand [B]; 42(10) 690-6 1988

Levels of ethene and propene, together with those of some other light hydrocarbons (propane, butane, isobutane and ethyne), have been measured under realistic conditions in environmental tobacco smoke (ETS) as a step towards the elucidation of the sources of 2-hydroxyethyl and 2-hydroxypropyl adducts of hemoglobin observed in non-smokers. These adducts may reflect in vivo doses of carcinogenic epoxides that are metabolites of the respective alkenes. The data show that 2.0 mg ethene, 1.4 mg propene, and 0.7 mg propane together with smaller amounts of butane, isobutane and ethyne are released per cigarette smoked (0.66 g tobacco) of a common Swedish brand. The alkenes in ETS should be considered as contributing factors to a risk of systemic cancer from passive smoking. With regard to alkene intake, even a relatively mild exposure to ETS (2 cigarettes per h for 5 h per day in a 33 m3 room with one air change per hour is estimated to correspond to the active smoking of about one cigarette per day.

109. MEASUREMENT OF NICOTINE IN BUILDING AIR AS AN INDICATOR OF TOBACCO SMOKE LEVELS

Williams DC, Whitaker JR, Jennings WG
Department of Food Science and Technology, University of California, Davis 95616.
Environ Health Perspect; 60:405-10 1985

Humans apparently differ greatly in their sensitivity and tolerance to tobacco smoke, thereby creating conflicts in the workplace. Resolution of conflicts in a large office complex at the authors' institution required an objective measure of smoke levels. A gas chromatographic technique was devised for collection and analysis of nicotine concentrations in the building air as an indicator of tobacco smoke pollution. Segregation of smokers and nonsmokers in the large office complex still resulted in substantial exposure of the nonsmoker to tobacco smoke, although a gradient of exposure was certainly observed. Passive tobacco smoke consumption in the smoking area of the office complex was calculated to be equivalent to 1.1 cigarettes per 8-hr period, and nicotine density in this area was 1.96 microgram/m. The restriction of smoking to a foyer area outside the office complex resulted in a slow but eventual reduction in nicotine concentrations in the office complex. Observed 'background' nicotine concentration levels corresponding to 4 to 7% of those encountered in smoking areas demonstrate that central air circulation systems and people movement increase the nicotine level throughout all rooms of a building, regardless of the smoking policies of an individual office complex. Recent documentation of the relationship between passive smoking and cancer, heart disease, pulmonary dysfunction, and allergic responses argues for restriction of smoking to building exteriors.

110. PASSIVE SMOKING AND DOMESTIC POLLUTION

Leuenberger P
Schweiz Rundsch Med Prax; 77(46):1254-6 1988

Keywords (MeSH): Adult; Carcinoma, Bronchogenic/etiology; Child; English Abstract; Human; Lung Neoplasms/etiology; Respiratory Function Tests; Respiratory Tract Diseases/

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etiology/physiopathology; Tobacco Smoke Pollution/*adverse effects/analysis.

111. ANALYTICAL CHEMICAL METHODS FOR THE DETECTION OF ENVIRONMENTAL TOBACCO SMOKE CONSTITUENTS (61 Refs)
Jenkins RA. Guerin MR
Bio. Organic Analysis Section. Oak Ridge National Laboratory. Tennessee.
Eur J Respir Dis Suppl. 133:33-40 1984

Keywords (MeSH): Aerosols; Aldehydes/analysis; Benzopyrenes/analysis; Carbon Monoxide/analysis; Carcinogens; Chromatography, Gas/instrumentation; Nicotine/analysis; Nitrogen Oxides/analysis; Nitrosamines/analysis; Review; Tobacco Smoke Pollution/*analysis.

112. ENVIRONMENTAL CARCINOGENS. METHODS OF ANALYSIS AND EXPOSURE MEASUREMENT. VOLUME 9 - PASSIVE SMOKING
Anonymous
IARC Sci Publ: (81):1-372 1987

Keywords (MeSH): Human; *Tobacco Smoke Pollution/adverse effects/analysis.

113. FORMATION AND PHYSICOCHEMICAL NATURE OF SIDESTREAM SMOKE (30 Refs)
Guerin MR
Analytical Chemistry Division. Oak Ridge National Laboratory. TN 37831.
IARC Sci Publ: (81):11-23 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Review; Review, Tutorial; Tobacco Smoke Pollution/*analysis.

114. INDOOR CONCENTRATIONS OF ENVIRONMENTAL TOBACCO SMOKE: MODELS DEALING WITH EFFECTS OF VENTILATION AND ROOM SIZE (32 Refs)
Repace JL
US Environmental Protection Agency. Office of Air and Radiation. Washington, DC 20460.
IARC Sci Publ: (81):25-41 1987

Keywords (MeSH): Air Pollutants, Environmental/analysis; Carbon Monoxide/analysis; Review; Review, Tutorial; Smoke/analysis; *Tobacco Smoke Pollution; *Ventilation.

115. GENERAL ANALYTICAL CONSIDERATIONS FOR THE SAMPLING OF TOBACCO SMOKE IN INDOOR AIR (19 Refs)
Jenkins RA. Guerin MR
Oak Ridge National Laboratory, TN 37831.
IARC Sci Publ: (81):105-13 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Review; Review, Tutorial; Tobacco Smoke Pollution/*analysis.

116. THE ANALYSIS OF THE PARTICULATE AND VAPOUR PHASES OF TOBACCO SMOKE (79 Refs)
Guerin MR. Higgins CE. Griest WH
Analytical Chemistry Division. Oak Ridge National Laboratory. TN 37831.
IARC Sci Publ: (81):115-39 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Chromatography, Gas; Review; Review, Tutorial; Spectrum Analysis, Mass; Tobacco Smoke Pollution/*analysis.

117. INDOOR CONCENTRATIONS OF ENVIRONMENTAL TOBACCO SMOKE: FIELD SURVEYS (43 Refs)
Repace JL
US Environmental Protection Agency. Office of Air and Radiation. Washington, DC 20460.
IARC Sci Publ: (81):141-62 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Environmental Exposure; Environmental Monitoring/*methods; Human; Review; Review, Tutorial; Tobacco Smoke Pollution; Ventilation.

118. A NEW DEVICE FOR THE GENERATION AND SAMPLING OF MAINSTREAM AND SIDESTREAM SMOKE AND FOR STUDIES OF SMOKE EXPOSURE
Griffith RB. Davis DL
Tobacco and Health Research Institute. University of Kentucky. Lexington 40546.
IARC Sci Publ: (81):163-74 1987

Keywords (MeSH): Carbon Monoxide/analysis; *Equipment Design; Nicotine/analysis; Smoke/*analysis; Tobacco/analysis.

119. SAMPLING AND ANALYSIS OF PHENOLS IN SIDESTREAM SMOKE AND INDOOR AIR (14 Refs)
Tomkins BA. Darrall KG. Zai ES. Phillips GF
Analytical Chemistry Division. Oak Ridge National Laboratory. TN 37831.
IARC Sci Publ: (81):175-81 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Chromatography, Gas; Chromatography, High Pressure Liquid; Phenols/*analysis; Review; Review, Tutorial; Tobacco Smoke Pollution/*analysis.

120. DETERMINATION OF CARBON MONOXIDE IN SIDESTREAM TOBACCO SMOKE
Adams JD. O'Mara-Adams KJ
American Health Foundation. Valhalla. NY 10595.
IARC Sci Publ: (81):185-90 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Carbon Monoxide/*analysis; Chromatography, Gas/*methods; Tobacco Smoke Pollution/*analysis.

121. VOLATILE ALDEHYDES IN SIDESTREAM TOBACCO SMOKE
Adams JD. Parent-Ermini AJ
American Health Foundation. Valhalla. NY 10595.
IARC Sci Publ: (81):205-12 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Aldehydes/*analysis; Chromatography, High Pressure Liquid/methods; Tobacco Smoke Pollution/*analysis.

122. VOLATILE ALDEHYDES IN TOBACCO SMOKE-POLLUTED INDOOR AIR
Jenkins RA
Organic Chemistry Section. Oak Ridge National Laboratory. TN 37831.
IARC Sci Publ: (81):213-20 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Aldehydes/*analysis; Chromatography, High Pressure Liquid/methods; Tobacco Smoke Pollution/*analysis.

123. VOLATILE N-NITROSAMINES IN SIDESTREAM SMOKE AND INDOOR AIR
Brunnemann KD
American Health Foundation. Valhalla. NY 10595.
IARC Sci Publ: (81):221-9 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Chromatography, Gas/methods; Nitrosamines/*analysis; Tobacco Smoke Pollution/*analysis.

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124. ANALYSIS OF TOBACCO-SPECIFIC NITROSAMINES IN SIDESTREAM TOBACCO SMOKE
Adams JD, Brunnemann KD
Ameican Health Foundation, Valhalla, NY 10595.
IARC Sci Publ; (81):231-7 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Chromatography, Gas/methods; Nitrosamines/*analysis; Tobacco Smoke Pollution/*analysis.

125. NICOTINE AND MINOR TOBACCO ALKALOIDS IN SIDESTREAM SMOKE AND INDOOR AIR
Brunnemann KD, Adams JD
American Health Foundation, Valhalla, New York 10595
IARC Sci Publ; (81):239-46 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Alkaloids/*analysis; Chromatography, Gas/methods; Nicotine/*analysis; Tobacco Smoke Pollution/*analysis.

126. GAS CHROMATOGRAPHIC DETERMINATION OF POLYCYCLIC AROMATIC HYDROCARBONS IN SIDESTREAM SMOKE AND INDOOR AIR
Grimmer G, Naujack KW
Biochemisches Institut fur Umweltcarcinogene, Grosshansdorf, FRG.
IARC Sci Publ; (81):249-68 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Chromatography, Gas/*methods; Polycyclic Hydrocarbons/*analysis; Tobacco Smoke Pollution/*analysis.

127. DETERMINATION OF AROMATIC AMINES IN SIDESTREAM SMOKE BY GAS CHROMATOGRAPHY
el-Bayoumy K, O'Donnell M, Hoffmann D
American Health Foundation, Valhalla, NY 10595.
IARC Sci Publ; (81):269-77 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Amines/*analysis; Chromatography, Gas/*methods; Tobacco Smoke Pollution/*analysis.

128. GAS CHROMATOGRAPHIC DETERMINATION OF PHENOLS IN SIDESTREAM SMOKE AND INDOOR AIR
Tomkins BA
Organic Chemistry Section, Oak Ridge National Laboratory, TN 37831.
IARC Sci Publ; (81):279-89 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Chromatography, Gas/*methods; Phenols/*analysis; Tobacco Smoke Pollution/*analysis.

129. II.2.B VOLATILE NITROSAMINES IN TOBACCO AND MAINSTREAM AND SIDESTREAM SMOKE AND INDOOR ENVIRONMENTS
Hoffmann D, Brunnemann KD, Webb KS
American Health Foundation, Valhalla, New York.
IARC Sci Publ; 45:69-83 1983

Keywords (MeSH): Carbon Radioisotopes/diagnostic use; *Climate; Environmental Pollution; Human; *Microclimate; Nitrosamines/*analysis; Radioisotope Dilution Technic; Smoke/*analysis; Smoking; Tobacco/*analysis.

130. II.2.C N-NITROSODIETHANOLAMINE IN TOBACCO AND MAINSTREAM AND SIDESTREAM SMOKE
Brunnemann KD, Hoffmann D
American Health Foundation, Valhalla, New York.
IARC Sci Publ; 45:85-92 1983

Keywords (MeSH): Carbon Radioisotopes; Carcinogens/*analysis; Diethylnitrosamine/analogs and derivatives/*analysis; Nitrosamines/*analysis; Radioisotope Dilution Technic; Smoke/*analysis; Tobacco/*analysis.

B.
Biological Monitoring of Environmental Tobacco Smoke Exposure

131. ENVIRONMENTAL TOBACCO SMOKE: OVERVIEW OF CHEMICAL COMPOSITION AND GENOTOXIC COMPONENTS
Lofroth G
Nordic School of Public Health, Gothenburg, Sweden
Mutat Res; 222(2):73-80 1989

Tobacco smoke contains numerous compounds emitted as gases and condensed tar particles. The sidestream smoke emissions, which constitute the major part of environmental tobacco smoke (ETS), are generally larger than the mainstream smoke emissions. Many of the organic compounds, belonging to a variety of chemical classes, are known to be genotoxic and carcinogenic. These include the known constituents, alkenes, nitrosamines, aromatic and heterocyclic hydrocarbons and amines. Emission of sidestream smoke in indoor environments with relatively low ventilation rates can result in pollutant concentrations above those generally encountered in ambient air in urban areas. The chemical characteristics of ETS thus support the indications that exposure to ETS can be causally associated with the induction of several types of cancer. (49 Refs)

132. PUBLIC EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE
Lofroth G, Ling PI, Agurell E
Nordic School of Public Health, Gothenburg, Sweden.
Mutat Res; 202(1):103-10 1988

Airborne particulate matter has been collected by personal samplers in public indoor areas and travel situations with environmental tobacco smoke pollution. Following extraction, the samples were assayed for mutagenicity in the presence of S9 with a sensitive microsuspension test using Salmonella TA98. The mutagenic responses of indoor air from public areas were much higher than those of ambient outdoor air. Depending on the circumstances, the mutagenic response varied in trains and airplanes but the results show that physical separation of non-smoking sections from smoking sections is necessary in order to achieve genuine non-smoking areas. Chemical fractionation and mutagenicity assay of the basic fraction show that Salmonella mutagenicity of airborne particulate matter might be used as a tobacco smoke-specific indicator, as the basic fraction of environmental tobacco smoke contains a large part of the mutagenic activity whereas this is not the case for outdoor ambient airborne particulate matter.

133. A GENOTOXIC ASSESSMENT OF ENVIRONMENTAL TOBACCO SMOKE USING BACTERIAL BIOASSAYS
Claxton LD, Morin RS, Hughes TJ, Lewtas J
Genetic Toxicology Division, U.S. Environmental Protection Agency, Research Triangle Park, NC 27711.
Mutat Res; 222(2):81-99 1989

Recently, the National Research Council in the U.S.A. stated that laboratory studies of environmental tobacco smoke (ETS) should be important in identifying ETS carcinogens, their concentrations in typical daily environments, and in understanding how these compounds contribute to ETS dose-response relationships. This paper demonstrates that integrated chemical and bacterial mutagenicity information can be used to identify ETS genotoxicants, monitor human exposure, and make comparative assessments. Approximately 1/3 of the ETS constituents for which there is quantitative analytical chemistry information also have associated genotoxicity information. For example, 11 of the quantitated compounds

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are animal carcinogens. Work presented in this paper demonstrates that both the nonparticle-bound semivolatile and the particulate-bound organic material contain bacterial mutagens. These ETS organics give an equivalent of approximately 86,000 revertants per cigarette. In addition, this article summarized efforts to estimate ETS bacterial mutagenicity, to use bacterial tests for the monitoring of ETS-impacted indoor environments, and to use bacterial assays for the direct monitoring of human exposure. (61 Refs)

134. ENVIRONMENTAL TOBACCO SMOKE: MUTAGENIC EMISSION RATES AND THEIR RELATIONSHIP TO OTHER EMISSION FACTORS

Leutas J, Williams K, Lofroth G, Hammond K, Leaderer B
Health Effects Res. Lab., EPA, Research Triangle Park, NC
Available from National Technical Information Service, Springfield, VA as NTIS/PB87-191516, 9 p., 1987.

The objective of this study was to evaluate the emission rates and exposure concentrations of mutagens, nicotine, and particles from cigarettes. Studies were conducted under controlled laboratory and chamber conditions as well as in personal residences. The mutagenicity of environmental tobacco smoke was evaluated in three bioassays using two strains of Salmonella typhimurium. Strain TA98 was used in the standard plate-incorporation and microsuspension histidine reversion assays, and strain TM677 in a microsuspension forward mutation assay. The mutagenicity, expressed either per ug particle or per ug nicotine, appeared to be a relatively constant factor that did not vary significantly between various cigarette brands. These data are being used to model the emissions of mutagens to predict mutagenic exposure concentrations under various conditions.

135. HUMAN EXPOSURE TO MUTAGENS FROM INDOOR COMBUSTION SOURCES,

Lewtas J, Claxton LD, Mumford JL
Health Effects Research Lab., Research Triangle Park, NC
Available from National Technical Information Service, Springfield, VA as NTIS/PB87-191615, 9 p., 1987.

The authors have measured human exposure to mutagens, using indoor medium-volume samplers and personal samplers, in targeted field studies of homes in the US. The combustion sources included in these studies were woodstoves, fireplaces, gas appliances, cooking, and tobacco smoking. These studies demonstrate that the presence of environmental tobacco smoke (ETS) consistently results in significantly higher human exposure to mutagens than outdoor air or non-smoking indoor spaces. The mutagenic emission rates from the other indoor combustion sources (eg, kerosene heaters), as determined in chamber studies, are more variable than ETS, and are dependent on the combustion source design and operation. Woodstoves and fireplaces result in higher outdoor concentrations of mutagens, which may indirectly influence the concentration of mutagens indoors.

136. ASSESSMENT OF PASSIVE AND TRANSPLACENTAL EXPOSURE TO TOBACCO SMOKE

Sorsa M, Husgafvel-Pursiainen K
Institute of Occupational Health, Helsinki, Finland.
IARC Sci Publ; (89):129-32 1988

Although tobacco smoke has been shown to be highly genotoxic in various experimental systems, most nonmolecular methods designed to assess exposure to mutagens are too insensitive to detect passive exposure to tobacco smoke. Biochemical markers of intake - cotinine and thiocyanates in body fluids - were shown to be elevated after occupational, passive or transplacental exposure to tobacco smoke, while no response was seen in the frequency of sister chromatid exchanges (SCE) in cultured blood lymphocytes. After occupational exposure to environmental tobacco smoke, the intake marker levels are generally less than 5% of the levels found in active smokers, while cord blood levels (representing fetal exposure) are at about the same level as in the mothers at the time of delivery.

137. PASSIVE SMOKING AT WORK: BIOCHEMICAL AND BIOLOGICAL MEASURES OF EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

Husgafvel-Pursiainen K, Sorsa M, Engstrom K, Einisto P
Institute of Occupational Health, Helsinki, Finland
Int Arch Occup Environ Health; 59(4):337-45 1987

Several biochemical and biological measures of tobacco smoke intake were used to evaluate exposure of restaurant personnel to environmental tobacco smoke as compared with active smokers and non-exposed non-smokers. All of the measured parameters - carboxyhaemoglobin (COHb), thiocyanate (SCN) and cotinine in plasma, cotinine and mutagenicity in urine, total white blood cell count (WBC), and sister chromatid exchange (SCE) frequency in cultured lymphocytes - were significantly elevated in the smoker group (n = 22) compared to the non-exposed group (n = 20). Work-related passive exposure (n = 27) was seen most clearly in the cotinine values, both from plasma (mean P-cot in passive smokers 10 ng/ml vs 5.2 ng/ml in non-exposed) and from urine (mean U-cot in passive smokers 56 ng/ml vs 8.3 ng/ml in non-exposed), but significant increases were also seen in the thiocyanate levels (mean P-SNC in passive smokers 58 mumol/l vs 46 mumol/l in non-exposed) and, as a preliminary finding, in total leucocyte count (in passive smokers 8.0 X 10(9)/l vs 6.8 X 10(9)/l in non-exposed). The results demonstrate that environmental tobacco smoke may be an occupational health hazard.

138. UPTAKE OF ENVIRONMENTAL TOBACCO SMOKE

Jarvis MJ
Addiction Research Unit, Institute of Psychiatry, Denmark Hill, London, UK.
IARC Sci Publ; (81):43-58 1987

This chapter has reviewed evidence of the uptake of smoke constituents by nonsmokers through exposure to environmental tobacco smoke. Although carbon monoxide absorption reflects an acute exposure, nicotine and its metabolite cotinine are the best markers currently available. Nicotine is found in measurable concentrations in the saliva and urine of most urban nonsmokers, and is present in higher concentrations in those with some recent exposure. But the short half-life of nicotine in the body means that it is best suited to quantifying exposure over a period of a few hours only. Cotinine, which has a half-life of about one day, has been shown to be a valid and sensitive marker of current daily-life exposure to environmental tobacco smoke. Estimating the magnitude of the passive smoking dose is difficult, and it is of doubtful validity to extrapolate from the uptake of one marker to that of another. Over a period when one cigarette-equivalent of carbon monoxide is absorbed, the dose of nicotine appears to be only between one-tenth and one-third of a cigarette-equivalent. Measures of tar deposition are not available, but nicotine is probably a better guide than carbon monoxide to the size of the tar dose. It seems unlikely that nonsmokers could absorb more than one or two mg of nicotine in a day, even if they spend the majority of their waking hours in heavily smoke-polluted atmospheres. Comparison of cotinine concentrations in urban nonsmokers and active smokers suggests that average nonsmokers may receive a dose of about 0.2 mg of nicotine per day. This is a preliminary estimate which depends on a number of assumptions and will be subject to revision as data accumulate on more

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representative samples. Finally, the confirmation that dose-response relationships exist between cotinine concentrations and self-reported passive smoking validates questionnaire measures of the degree of environmental smoke exposure. Epidemiological studies which suggest that passive smoking carries a risk to health thereby gain increased credibility. But future progress in understanding will be best assured if epidemiological methods and biological monitoring of exposure markers are combined in the same studies. (37 Refs)

139. SIGNIFICANCE OF EXPOSURE TO SIDESTREAM TOBACCO SMOKE

Hoffmann D, Hoffmann I
American Health Foundation, Valhalla, NY 10595
IARC Sci Publ; (81):3-10 1987

The presence of toxins and carcinogens in ambient air polluted with tobacco smoke is largely due to the sidestream smoke emissions from the smouldering tobacco products. Levels of these toxins and carcinogens in sidestream smoke often exceed their concentrations in mainstream smoke. Dosimetry of tobacco-specific markers of exposure in physiologic fluids suggests that in regard to nicotine - which is the major tobacco alkaloid - exposure of humans to environmental tobacco smoke causes but a few percent of the nicotine levels reached as a result of active inhalation of cigarette mainstream smoke. Yet, this measurement of exposure is not universally applicable to all of the tobacco smoke pollutants in this complex matrix. Existing knowledge of the chemical composition of sidestream smoke and evidence of biological activity of sidestream smoke components suggests that this environmental pollutant has carcinogenic potential. Significance of exposure to environmental tobacco smoke must be evaluated on the basis of the severity of the pollution, the duration of exposure and personal variations in uptake. (36 Refs)

140. IARC APPROACHES TO MONITORING EXPOSURE OF PASSIVE SMOKING

O'Neill I, Riboli E
International Agency for Research on Cancer, Lyon, France.
Toxicol Lett; 35(1):29-33 1987

Existing information on the nature of passive smoking shows many pitfalls in ascertaining carcinogenic exposures. Two current activities of the International Agency for Research on Cancer are described: presentation of standardized monitoring methods for passive smoking; and an international study to explore biochemical measurements in relation to passive smoking history as assessed by interview.

141. PERSONAL, BIOLOGICAL AND AIR MONITORING FOR EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE (97 Refs)

Wallace LA, O'Neill IK
Harvard University School of Public Health, Boston, MA 02115.
IARC Sci Publ; (81):87-103 1987

Keywords (MeSH): Air Pollutants, Environmental/*analysis; Cotinine/urine; Environmental Monitoring/*instrumentation/methods; Human; Nicotine/urine; Review; Review, Tutorial; Tobacco Smoke Pollution/*analysis.

142. A CRITICAL LOOK AT N-NITROSAMINES IN ENVIRONMENTAL TOBACCO SMOKE

Hoffmann D, Adams JD, Brunnemann KD
Naylor Dana Institute for Disease Prevention, American Health Foundation, Valhalla, NY 10595.
Toxicol Lett; 35(1):1-8 1987

The amounts of cigarette smoke carcinogens released into the environment as sidestream smoke (SS) constituents are generally twice as high as the levels of the carcinogens that are inhaled as mainstream smoke (MS). However, certain carcinogens in undiluted SS, such as nitrosamines, exceed MS levels up to 50 times. Regardless of the fact that SS is usually substantially diluted before being inhaled, its constituents can be determined in environmental tobacco smoke (ETS) by modern analytical methods and levels of exposure of involuntary smokers can be assessed by the use of specific markers. Currently, the uptake of ETS by nonsmokers is determined by measuring nicotine and its metabolite cotinine in saliva, serum and/or urine. In on-going studies, we are now exploring the determination of the two highly carcinogenic nicotine-derived nitrosamines N'-nitrosonornicotine (NNN) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), respectively of their metabolites, in physiological fluids of smokers and nonsmokers.

143. MONITORING ABSORPTION BY MEANS OF DETERMINATION OF NICOTINE AND COTININE

Curtall M, Enzell CR
Research Department, Swedish Tobacco Company, Stockholm.
Arch Toxicol Suppl; 9:88-102 1986

Recent studies indicate that nonsmokers exposed to environmental tobacco smoke (ETS) acquire an impaired lung function and run an increased risk of lung cancer. These findings have been questioned mainly on the basis that the amount of ETS received by the nonsmoker has remained unquantified. The factural risk of ETS exposure could possibly be estimated by measuring the absorption of tobacco smoke products by nonsmokers. Nicotine, which is specific to tobacco, and its main metabolite, cotinine, have been used for this purpose. The usefulness of these markers based on data obtained for different body fluids from nonsmokers exposed to ETS under experimental as well as field conditions is discussed.

144. UPTAKE OF TOBACCO SMOKE COMPONENTS

Haley NJ, Hoffmann D, Wynder EL
Naylor Dana Inst. for Disease Prevention, American Health Foundation, Valhalla, NY 10595
Banbury Rep; 23:3-19 1986

Evaluation of tobacco smoke intake is discussed with respect to validation of self-reported smoking behavior, compensation and nicotine tolerance, transplacental exposure and fetal uptake, and passive smoking. The quantitation of cotinine, the major metabolite of nicotine, has proven quite useful in validating smoking behavior, especially in adolescents and announced quitters where daily use might be low, and in investigating mechanisms of smoker compensation. The presence of high levels of cotinine (greater than 200 ng/ml) in saliva, urine or plasma of adolescents suggests the use of smokeless tobacco since, to achieve such levels with cigarettes, the smoker would typically smoke at least 15 cigarettes each day. Such smoking behavior would result in greatly elevated thiocyanate levels; high cotinine in the presence of low thiocyanate, therefore, is associated with the use of chewing tobacco or oral snuff. Biochemical markers of smoke intake, such as cotinine in urine, are sufficiently sensitive and specific to identify passive smokers. A dose-response relationship for cotinine can be seen within 2-4 hr after exposure. Individuals evaluated under controlled conditions and in free-living situations generally have cotinine levels 0.1% and 2%, respectively, of those observed in cigarette smokers. A study of infants born to smoking and non-smoking mothers found that urine cotinine excretion was significantly greater in neonates of smoking mothers. However, among neonates of smokers, there was no clear relationship between newborn urine cotinine:creatinine ratio and maternal cigarette con-

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sumption during the 24 hr before delivery. The precise quantitative relationship between the measured levels of tobacco smoke markers and the intake of carcinogenic compounds in tobacco smoke is not known. The chapter concludes with a discussion by conference participants. (39 Refs)

145. TOBACCO SIDESTREAM SMOKE: UPTAKE BY NONSMOKERS

Hoffmann D Haley NJ. Adams JD. Brunnemann KD
Naylor Dana Institute for Disease Prevention. American Health Foundation. Valhalla. New York 10595.
Prev Med: 13(6) 608-17 1984

Some epidemiological studies indicate an association between passive smoking and an increased risk for cancer, especially for cancer of the lung. Other reports, however, have failed to confirm these findings. Biochemical analyses of the physiological fluids for markers of exposure to tobacco smoke are needed as measurements of the uptake of smoke components by nonsmokers and for the estimation of relative cancer risk to passively exposed persons compared with that to active cigarette smokers. This communication reports the uptake of carbon monoxide, hydrogen cyanide, and nicotine after passive smoke exposure under controlled conditions. The results indicate that salivary nicotine values reflect the level of recent passive smoke exposure within an hour and that urinary cotinine values indicate the level of passive smoke exposure in the preceding hours. N-Nitrosoproline has been shown to serve as an indicator of endogenous N-nitrosamine formation in cigarette smokers: yet, preliminary studies do not indicate that urinary excretion of N-nitrosoproline is increased following short-term passive smoke exposure. In infants, first field studies suggest a correlation between exposure to tobacco-smoke-polluted environments and levels of cotinine in both serum and urine.

146. EFFECTS OF ENVIRONMENTAL TOBACCO SMOKE ON URINARY COTININE EXCRETION IN NONSMOKERS. EVIDENCE FOR PASSIVE SMOKING

Matsukura S. Taminato T. Kitano N. Seino Y. Hamada H. Uchihashi M. Nakajima H. Hirata Y
N Engl J Med: 311(13):828-32 1984

Despite recognition of the deleterious effects of passive smoking, quantitative information on the intake of environmental tobacco smoke is still lacking. Cotinine is the major metabolite of nicotine found in the urine. We have examined the relationship between urinary cotinine excretion in 472 nonsmokers and the smokiness of their environment. The urinary cotinine levels of nonsmokers who lived with smokers were higher than those of nonsmokers who did not, increasing with the combined daily cigarette consumption of smokers in the family. The urinary cotinine values of nonsmokers who worked with smokers were also higher than those of nonsmokers who did not, increasing with the number of smokers in the workroom. The presence of smokers in both the home and the workplace also increased the cotinine levels. Urban nonsmokers had more cotinine in their urine than rural nonsmokers. We conclude that the deleterious effects of passive smoking may occur in proportion to the exposure of nonsmokers to smokers in the home, the workplace, and the community.

147. URINARY COTININE AS MARKER OF BREATHING OTHER PEOPLE'S TOBACCO SMOKE [LETTER]

Wald NJ. Boreham J. Bailey A. Ritchie C. Haddow JE. Knight G
Lancet: 1(8370):230-1 1984

148. DNA ADDUCTS, PROTEIN ADDUCTS, AND SISTER CHROMATID EXCHANGE IN CIGARETTE SMOKERS AND NONSMOKERS

Perera FP. Santella RM. Brenner D. Poirier MC. Munshi AA. Fischman HK. Van Ryzin J
Division of Environmental Sciences. Columbia University School of Public Health. New York. NY 10032.
J Natl Cancer Inst: 79(3):449-56 1987

In order to validate markers of internal dose and biologically effective dose of carcinogens, a battery of measurements was made on blood samples from 22 smokers and 24 nonsmokers. The markers included immunoreactivity in an enzyme-linked immunosorbent assay (ELISA) quantified in white blood cells with the use of a polyclonal anti-benzo[a]pyrene diol epoxide-I-DNA antibody, 4-aminobiphenyl hemoglobin (4-ABP-Hb) adducts measured by negative chemical ionization mass spectrometry, sister chromatid exchange (SCE) in cultured lymphocytes, and cotinine in plasma measured by radioimmunoassay. Several blood samples were drawn from each subject. In blood samples 1 and 3 having detectable levels of DNA adducts, mean femtomole-per-microgram levels were consistently higher among smokers compared to nonsmokers. The borderline significance of this difference may be attributable to the small numbers of subjects. Consistently higher adduct levels were seen in females compared to males. In sample 3, adduct levels were significantly correlated with measurements of active smoking in smokers and with passive smoking in nonsmokers. By contrast to the ELISA data, which may reflect cumulative exposure from multiple background sources, the 4-ABP-Hb assay was able to distinguish clearly between smokers and nonsmokers. SCEs were significantly elevated in the smokers compared to nonsmokers. Also observed were significant correlations between 4-ABP-Hb and both cotinine and SCEs, as well as a positive correlation between the 4-ABP-Hb and DNA adduct levels (sample 3) that was highly significant. The correlation between DNA and 4-ABP-Hb adducts was significant in smokers but not nonsmokers (sample 3). These results support the need for batteries of markers to detect and to quantify the carcinogenic dose to humans resulting from both specific and 'background' environmental exposures.

149. COLLECTION OF URINE FOR PROSPECTIVE STUDIES IN PASSIVE SMOKING

Haley NJ, O'Neill IK
Section of Clinical Biochemistry, American Health Foundation, Valhalla, NY 10595.
IARC Sci Publ; (81):293-7 1987

Keywords (MeSH): Human; Prospective Studies; *Tobacco Smoke Pollution; *Urine.

150. QUESTIONNAIRE USED IN THE 'INTERNATIONAL STUDY ON EXPOSURE TO OTHER PEOPLE'S SMOKE AND URINARY COTININE LEVELS IN NONSMOKERS'

Riboli E
International Agency for Research on Cancer, Unit of Analytical Epidemiology, Lyon, France.
IARC Sci Publ; (81):353-72 1987

Keywords (MeSH): Cotinine/*urine; Data Collection; Female; Human; Middle Age; Pyrrolidinones/*urine; Questionnaires; *Tobacco Smoke Pollution.

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IV.

REVIEWS ON THE HEALTH EFFECT OF PASSIVE SMOKING

151. THE PERILS OF SECOND-HAND SMOKING

Stock S
No affiliation given
Neu Sci. 88(1221):10-13 1980

A cigarette produces approx 2 times as much sidestream smoke (SS) (into the air) as mainstream smoke (MS) (drawn into the mouth). Many chemicals in tobacco smoke are present in higher concentrations in SS. SS is unfiltered by the cigarette and by the smoker himself. It is possible that passive smoking of SS could yield the same spectrum of disease for others as suffered by the smoker himself. There are only a few studies of the effects of passive smoking. At least 50% of nonsmokers report definite ill effects from passive smoking. People with allergic disease are more likely to suffer symptoms when exposed to cigarette smoke. Tobacco smoke contains approx 1,000 polycyclic aromatic hydrocarbons, and various other carcinogenic substances. Nonsmokers in a smoky room may inhale these compounds in quantities equivalent to smoking several cigarettes. Nonsmokers who work in smoky offices have been found to have Cushman's spirals in their sputum, while those who are not exposed, do not. The lung function of passive smokers has been shown to be impaired. A cigarette generates nicotine and carbon monoxide into SS. Children of parents who smoke have more lower and upper respiratory tract disease than those of nonsmoking parents. Wives of smokers die an av of 4 yr earlier than wives of nonsmokers. Tobacco smoke removes negative ions from the air. Smoke can probably sensitize a person to a glycoprotein, which may accelerate the development of atherosclerosis. (no Refs)

152. THE HEALTH CONSEQUENCES OF SMOKING. CANCER. A REPORT OF THE SURGEON GENERAL

Luoto J, Shopland DR
Office on Smoking and Health, Public Health Service, United States Dept. Health and Human Services, Rockville, MD. 20857
The Health Consequences of Smoking. Cancer. A Report of the Surgeon General. Washington, D. C., NIH Publication No. 82-50179, 322 pp., 1982.

The 1982 Report on the Health Consequences of Smoking, prepared in accordance with the Public Health Cigarette Smoking Act of 1969 and the Federal Cigarette Labeling and Advertising Act is presented focusing on a comprehensive assessment of the associations reported between smoking and various cancers. Biomedical evidence for determining causality between smoking and cancer, mechanisms of carcinogenesis, connection between involuntary smoking and lung cancer, and cessation of smoking are discussed. Smoking has been implicated as a cause of cancer of the lung, larynx, oral cavity, and esophagus, and associated with cancer of the urinary bladder, kidney, and pancreas. Provisional mortality data for 1980 indicate that cancer was responsible for approx 412,000 deaths in the United States. Investigations have suggested that 22-38% of these deaths can be attributed to smoking, and therefore are potentially 'avoidable' if smoking did not exist as human behavior. With increasing duration of smoking cessation, overall cancer death rates decline, approaching the death rate of nonsmokers. Lung cancer mortality increases with increasing dosage of smoke exposure and is inversely related to age of initiation. The biological activity of whole cigarette smoke and its tar and tar fractions can be measured by improved inhalation assays and tests for tumor-initiating, tumor-promoting, and cocarcinogenic activities on mouse skin. (759 Refs)

153. IMPORTANT NEW FINDINGS ON PASSIVE SMOKING

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Off Gesundheitswes. 44(2):130-132 1982

Five arguments for defining passive smoking as a true health risk, rather than merely an annoyance, are presented. Tobacco smoke contains greater than 40 carcinogenic substances. Most of these substances are not inhaled by the active smoker alone. Carcinogenicity of the substances has been confirmed in animal studies. Carcinogens in smoke show a summation effect without evidence of a threshold effect. Recent studies are cited to support arguments for imposition of legal restrictions on smoking in public. (35 Refs)

154. PASSIVE SMOKING-A HEALTH HAZARD?

Weber A
Institut fur Hygiene und Arbeitsphysiologie. Eidgenossischen Technischen Hochschule. Clausiusstr. 21. CH-8092 Zurich. Switzerland
Dtsch Med Wochenschr: 107(9):323-325 1982

The question of whether passive smoking poses a health hazard is addressed. The following topics are considered dangers of passive smoking for children and sick people, especially those with asthma, chronic bronchitis, etc; evidence to support and refute the contention that passive smoking increases the risk of development of lung cancer; lung function in passive smokers in the workplace; and passive smoking and subjective evaluation of its effects. Recommended levels of exposure and measures for reducing the amount of passive smoking are discussed. (28 Refs)

155. NO PROOF OF HEALTH HAZARD

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Institut fur Klinische Chemie. Deutsches Herzzentrum. Lothstr. 11. D-8000 Munchen 2. W. Germany
MMW: 124(4):13-15 1982

Health hazards of passive smoking (PS) are reviewed. PS presents an inconvenience for a healthy adult, but no evidence of a real hazard for health was yet presented. Forty carcinogenic components could be determined in cigarette smoke, the majority of which are released into the ambient atmosphere and could be inhaled by the passive smoker. Nitrosamines deserve special attention.

156. THE HEALTH EFFECTS OF INVOLUNTARY SMOKING

Weiss ST. Tager IB. Schenker M. Speizer FE
Channing Laboratory. Brigham and Women's Hospital.
Am Rev Respir Dis: 128(5):933-42 1983

Involuntary smoking is defined as the exposure of nonsmokers to tobacco combustion products in the indoor environment. Involuntary smokers are exposed to a quantitatively smaller and qualitatively different smoke exposure than active smokers. Quantitation of exposure is particularly difficult in both physiologic and epidemiologic studies. Acute physiologic studies have documented minimal physiologic changes in healthy subjects. However, individuals with heart or lung disease may be differentially affected. A relatively large body of data relates parental (particularly maternal) cigarette smoking to the occurrence of both acute respiratory illnesses and chronic respiratory symptoms in children. The effect seems to be greatest early in life and cannot be separated from in utero exposure. Data linking parental smoking

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to lower levels of pulmonary function are all cross-sectional and less conclusive. What is apparent is that the magnitude of the direct effect of passive smoke exposure is likely to be relatively small (from 1 to 5% reduction in maximally obtained lung function level in exposed children). Data on adults are insufficient to allow for a quantitative estimate. The important effects of passive smoke exposure in childhood are twofold. The slight reduction in pulmonary function level may predispose individuals to increased risks from environmental agents later in life. In addition, having a parent who smokes substantially increases the likelihood that a child will become a smoker. Finally, two studies have linked lung cancer in nonsmokers to exposure to spouses' cigarette smoke. Further research is needed to confirm these findings. Involuntary smoking may have important health effects, either direct or indirect, which deserve further study.

157. INDOOR AIR POLLUTION: A PUBLIC HEALTH PERSPECTIVE

Spengler JD, Sexton K
Department of Environmental Science and Physiology, Harvard University School of Public Health, Boston, Massachusetts.
Science; 221(4605):9-17 1983

Although official efforts to control air pollution have traditionally focused on outdoor air, it is now apparent that elevated contaminant concentrations are common inside some private and public buildings. Concerns about potential public health problems due to indoor air pollution are based on evidence that urban residents typically spend more than 90 percent of their time indoors, concentrations of some contaminants are higher indoors than outdoors, and for some pollutants personal exposures are not characterized adequately by outdoor measurements. Among the more important indoor contaminants associated with health or irritation effects are passive tobacco smoke, radon decay products, carbon monoxide, nitrogen dioxide, formaldehyde, asbestos fibers, microorganisms, and aeroallergens. Efforts to assess health risks associated with indoor air pollution are limited by insufficient information about the number of people exposed, the pattern and severity of exposures, and the health consequences of exposures. An overall strategy should be developed to investigate indoor exposures, health effects, control options, and public policy alternatives.

158. CANCERS FROM TOBACCO SMOKE

Castot A, Efthymiou ML
Centre de Toxicologie, Hopital Fernand Widal, Paris.
Sem Hop Paris; 59(27-28):[illegible]-30 1983

The proximity of smokers exposes non-smokers to the inhalation of tobacco smoke. The existence of this absorption has been established by assays of blood nicotine concentrations or urinary thiocyanate concentrations, in volunteers: proximity with an individual who smokes approximately ten cigarettes is equivalent to smoking one cigarette. Consequences of this passive smoking are not negligible. Two studies, one Japanese and one Greek, have shown that the risk of lung cancer in wives of smokers is twice that in non-smoking women married to non-smokers, even though various American studies seem more reassuring. Another risk of cancer exists in the offspring of mothers who smoked during their pregnancy, with a rate of 14.9 x 10(5) if the mother smoked, versus 11. 4 x 10(5) if she didn't. Experimental rates of cancer in hamster fetuses confirm this risk.

159. GENOTOXIC RISK OF PASSIVE SMOKING

Bos RP, Henderson PT
Institute of Pharmacology, University of Nijmegen, The Netherlands.
Rev Environ Health; 4(2):161-78 1984

More than 60 chemical components are identified in cigarette smoke which have shown to be carcinogenic. The presence of these chemicals is established in mainstream smoke. However, many of them also appear in sidestream smoke resulting in pollution of indoor air, as is shown by the presence of mutagenic substances. Some rather potent carcinogens like N-nitroso-dimethylamine and benzo(a)pyrene have been established in the air of smoke filled rooms. Only a few studies describe internal exposure of passive smokers. Deposition of sidestream smoke in the human respiratory tract has been established for passive smokers. On the other hand, it was shown that inhalation of air contaminated with sidestream smoke results in an increase in the urinary excretion of products mutagenic in the Salmonella/microsome assay. Three epidemiological studies showed an increased risk of lung cancer for non-smoking wives having smoking husbands. Since it is generally acknowledged that most of the genotoxic carcinogens can be detected by in vitro mutagenicity tests, mutagenicity in urine of passive smokers can be considered as an indication of exposure to carcinogens. This observation suggests that there is a causality in the association between increased cancer risk and passive smoking as was found in three epidemiological studies. It is generally accepted that genotoxic chemicals exert their effects in direct proportion to the level of exposure, which means that for these agents no safe thresholds can be established. Several studies clearly show the presence of genotoxic substances in indoor air as a consequence of smoking. Therefore, the outcome of the epidemiological studies is not surprising. As long as half of the human population persists in smoking, the problems of involuntary inhalation of genotoxic substances will continue for the other half. Strategies to control the environmental cancer problem can only be successful if the health hazards of passive smoking are taken seriously. (46 Refs)

160. PASSIVE SMOKING

Lee PN
Independent Consultant in Statistics and Epidemiology, 25 Cedar Road, Sutton, Surrey, England
Ettore Majorana Int Sci Ser Life Sci; 17:187-214 1984

Passive smoking can be defined as the inhalation of tobacco smoke other than by puffing on a cigarette, cigar, or pipe. Passive smoking is discussed under the following headings: early claims (annoyance and irritation; allergy; respiratory effects in children; psychomotor effects; exercise tolerance), dosimetry (general; nicotine; total particulate matter; carbon monoxide; N-nitrosodimethylamine; other considerations for epidemiological interpretation), effects on the small airways, lung cancer, and total mortality. Review of dosimetric evidence for passive smoking suggests that exposure of non-smokers in average circumstances from passive smoke will be substantially less than exposure due to smoking cigarettes actively. The difference in exposure is increased when taking into consideration that, in smokers, passive exposure to their own smoke doubtless exceeds the passive exposure they pass on to non-smokers. Studies on passive smoking are reviewed, including the Japanese, Greek, United States, Hong Kong, Louisiana, and German studies. Much of the data presented suggesting that passive smoking is associated with more serious health hazards than previously thought is open to considerable criticism, and none of the data are by any means completely convincing. More research is certainly needed, but at present the view that passive smoking does not result in any

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material risk of serious disease for the healthy non-smoker remains a reasonable one. (38 Refs)

161. BREATHING OTHER PEOPLE'S SMOKE

Horsfield K
Midhurst Medical Res. Inst., Midhurst, West Sussex, GU29 0BL, England
Ettore Majorana Int Sci Ser Life Sci: 17:169-83 1984

Some of the health consequences of active and passive smoking are reviewed, and the difficulties in studying the effects of passive smoking are examined. Possible health consequences of smoking and of breathing other people's smoke are listed in a table. Cancer of the lung is the only cancer reported to be associated with passive smoking. Evidence suggesting a causative link between passive smoking and lung cancer has not been very convincing, but additional evidence in support of the contention is gradually being obtained. People with ischemic heart disease may be adversely affected by passive smoking. Patients suffering from chronic bronchitis, emphysema, and chronic obstructive lung disease also may be affected by passive smoking. Lung infections are more common in smokers, and young children whose parents smoke are twice as likely to develop acute bronchitis or pneumonia as those whose parents do not smoke. Occasionally an attack of wheezing may be precipitated in an asthmatic by passive smoking. Many people suffer annoyance and discomfort when exposed to other people's smoke, and this may be true of smokers as well as of non-smokers. Symptoms in those who breath other people's smoke include eye irritation, watering, blinking, rubbing, nose and throat irritation, cough, headache, smell of smoke, smell on clothes, and smell in room afterwards. Cigarette smoke contains over 2,000 compounds, many of which are potentially poisonous or carcinogenic. Various kinds of smoke, of very variable composition, mix with ambient air and are diluted to a variable degree; both the active and the passive smoker breath the resulting smoky air, but the smoker, being nearer to the source of pollution, takes in much more smoke passively than does the non-smoker. Absorption of smoke by the passive smoker depends on the levels reaching him/her in the ambient air and on the duration of exposure. Because of the number of variables and difficulties encountered, there is no satisfactory way of measuring passive smoking. (9 Refs)

162. LUNG DISEASES IN THE ELDERLY. PATHOGENETIC SIGNIFICANCE OF POLLUTANTS AND ENVIRONMENTAL FACTORS

Heitz M, Herzog H
Abteilung fur Atmungskrankheiten, Universitat Basel.
Fortschr Med; 102(17):477-82 1984

In the elderly pneumoconioses due to anorganic or organic dusts are not very common. The incidence of mesothelioma is increasing also in the elderly population. Mesothelioma has become the most frequent occupational malignancy. There is also evidence that mesothelioma can be produced by other fibers than asbestos particles. The paper discusses further the effect of passive smoking, where there is new evidence that passive-smokers are exposed to a higher risk for bronchial cancer than non-smokers. The interactions between smoking and air pollution and morbidity of chronic bronchitis are illustrated. New aspects of pathogenesis and pathophysiology of chronic obstructive lung disease and pulmonary emphysema due to smoking as the most frequent environmental lung disease in the elderly are further discussed. A brief overview of the therapeutical approach to chronic obstructive lung disease including new forms of treatment of cor pulmonale is finally given.

163. SMOKING AND THE LUNG

Anonymous
No affiliation given
Ettore Majorana Int Sci Ser Life Sci: 17:1-504 1984

Reports by experts from various parts of the world on the effects of cigarette smoking on the lung are presented under the following headings: structural aspects of cigarette smoke-induced pulmonary disease; the reflex effects of cigarette smoking; carotid body hyperplasia; cocarcinogenic effect of tobacco smoke in rats; the act of smoking; the quantitation of smoke uptake; physiological effects of changing cigarette nicotine yield; proteases and antiproteases in the normal lower respiratory tract; smoke-induced modification of alveolar macrophage activity; tobacco smoke and alveolar cell populations; proteases - antiproteases in the lower respiratory tract of patients with alpha 1-antitrypsin deficiency and cigarette smokers; breathing other people's smoke; passive smoke; nicotine and the control of smoking behavior; work and stress as motives for smoking; particulate deposition in smoking; elastin-lysine derived cross-links structure, biosynthesis and relation to lung emphysema pathogenesis; effects of cigarette smoke on the metabolism of arachidonic acid and prostaglandins in lung; consequences of smoking and smokers at risk; ciliary activity in the respiratory tract and the effects of tobacco smoke; the gas phase of tobacco smoke and the development of lung diseases; changes in lung function after smoking cessation (interrelationship between smoking, lung function and body wt); chronic bronchitis and decline in pulmonary function with some suggestions on terminology; does smoking have any influence on the course of restrictive lung disease; the single breath nitrogen test; the relation between increased bronchial reactivity and annual decline in airway function in smokers; and a comparison of the ability of different lung function tests to discriminate asymptomatic smokers and non-smokers.

164. ENVIRONMENTAL TOBACCO SMOKE AND LUNG CANCER (17 Refs)

Rylander R
Department of Environmental Hygiene, University of Gothenburg, Sweden.
Eur J Respir Dis Suppl: 133:127-33 1984

Keywords (MeSH): Adult; Female; Human; Lung Neoplasms/*etiology; Male; Marriage; Review; Tobacco Smoke Pollution/*adverse effects.

165. ROUNDTABLE DISCUSSION: SYMPOSIUM: MEDICAL PERSPECTIVES ON PASSIVE SMOKING

Lehnert G, Garfinkel L, Hirayama T, Schmahl D, Uberla K, Wynder EL, Lee P
Zentralinstitut fur Arbeitsmedizin, Hamburg, West Germany.
Prev Med: 13(6):730-46 1984

Keywords (MeSH): Adenocarcinoma/etiology; Age Factors; Air Pollutants, Occupational/adverse effects; Air Pollution/adverse effects; Animal; Dogs; Dose-Response Relationship, Drug; Environmental Exposure; Epidemiologic Methods; Female; Human; Lung Neoplasms/*etiology; Male; Marriage; Neoplasms/etiology; Rats; Risk; Smoking; Time Factors; Tobacco Smoke Pollution/*adverse effects.

166. ARE ACTIVE AND PASSIVE SMOKING HARMFUL? DETERMINING CAUSATION

Guyatt GH, Newhouse MT
McMaster University, Hamilton, Ontario, Canada.
Chest; 88(3):445-51 1985

Assessing the evidence regarding any causal question involves examining the strength of the studies conducted and applying a series of 'diagnostic tests' for causation. We have

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reviewed the strength of the evidence incriminating smoking as a cause of lung cancer, and passive smoking as a cause of respiratory illness and decreased pulmonary function in children. There are eight prospective studies of smoking and lung cancer which have consistently shown a strong relationship. These studies have confirmed the temporality of the association and demonstrated a dose-response gradient. The studies addressing the effects of passive smoking in children are considerably weaker. Although they are consistent in suggesting increased infections for children less than one year of age, neither increased risk nor a dose-response gradient is consistently found in older children and the effect size, when present, is small. The rules for assessing causation applied here can be used to integrate new information concerning the health hazards of smoking.

167. INDOOR AIR POLLUTION

Spengler JD

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N Engl Reg Allergy Proc; 6(2):126-34 1985

Although official efforts to control air pollution have traditionally focused on outdoor air, it is now apparent that elevated contaminant concentrations are common inside some private and public buildings. Concerns about potential public health problems due to indoor air pollution are based on evidence that urban residents typically spend more than 90 percent of their time indoors, concentrations of some contaminants are higher indoors than outdoors, and for some pollutants personal exposures are not characterized adequately by outdoor measurements. Among the more important indoor contaminants associated with health or irritation effects are passive tobacco smoke, radon decay products, carbon monoxide, nitrogen dioxide, formaldehyde, asbestos fibers, microorganisms and aeroallergens. Efforts to assess health risks associated with indoor air pollution are limited by insufficient information about the number of people exposed, the pattern and severity of exposures, and the health consequences of exposures. An overall strategy should be developed to investigate indoor exposures, health effects, control options, and public policy alternatives.

168. CANCER RISKS: SMOKING

Wald NJ

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Cancer Risks and Prevention. Vessey MP, Gray M, eds. New York. Oxford University Press, p. 44-67, 1985.

Cigarette smoking is established as the principal cause of lung cancer in Western countries, and it is also becoming the leading cause of lung cancer in an increasing number of economically less-developed countries. Although the basic mechanism by which a normal cell becomes malignant is still unknown, epidemiological enquiry has yielded comprehensive information on the dose-response relationship between the amount smoked and the risk of lung cancer, on the effects of duration of exposure assessed independently of the amount smoked, and on the interaction with other pulmonary carcinogens such as asbestos and ionizing radiation. Smoking is discussed under the following headings: trends in cigarette smoking in Great Britain; trends in lung cancer mortality in England and Wales; trends in coronary heart disease mortality in England and Wales; what component of cigarette smoke causes coronary heart disease; do people smoke to obtain nicotine; compensatory smoking; the inhaling anomaly; and breathing other people's smoke. Cigarette smoking is a habit which is now declining steadily in Great Britain. The reduction in tar yields and the reduction in cigarette smoking have produced a notable decline in lung cancer. Coronary heart disease, however, has not changed in the same way. Biochemical studies on compensatory smoking suggest that the intake of smoke components, particularly nicotine and carbon monoxide, has not decreased as much as had previously been expected, and this may explain the failure to observe an effect of low tar cigarettes on coronary heart disease mortality. Recent data on biochemical estimates of exposure to other people's smoke predict a risk of lung cancer that is of similar order of magnitude to the risk observed in non-smokers living with smoking spouses. The risk is small, but nonetheless real, and places smoking in a category comparable to other environmental and occupational carcinogens. (25 Refs)

169. LUNG CANCER ETIOLOGY: CHALLENGES OF THE FUTURE

Wynder EL, Goodman MT, Hoffmann D

American Health Foundation, New York, New York 10017.

Carcinog Compr Surv; 8:39-62 1985

The 1982 Report of the Surgeon General of the U.S. Public Health Service concluded that 'cigarette smoking is the major single cause of cancer mortality in the United States' and that '85 percent of lung cancer cases are due to smoking'. Thus, major emphasis should be placed on school health education programs designed to prevent young people from smoking. Those students who are already cigarette smokers should be provided with an opportunity to attend smoking cessation courses with the hope that they stop. However, as long as society condones tobacco usage, millions of people will smoke, and millions of others will be involuntarily exposed to tobacco smoke. In this communication we have discussed the need for future research on the etiology of lung cancer. This includes the observation of a shift toward an increasing proportion of adenocarcinoma compared to squamous cell carcinoma of the lung in men, more detailed knowledge of the effects of macro- and micronutrients in the etiology of lung cancer, a clear delineation of the impact of tumor initiators, tumor promoters, and cocarcinogens in the development of lung cancer in cigarette smokers, and a study of the effects of the low-yield cigarette on the lung cancer risk of smokers. Finally, we reviewed the present knowledge as to the possible association of passive smoke exposure and lung cancer. Here we have placed major emphasis on the need for a close cooperation between epidemiologists and clinical biochemists in risk assessment.

170. CIGARETTE SMOKING AND HEALTH

Mason RJ, Buist AS, Fisher EB, Merchant JA, Samet JM, Welsh CH

Am Rev Respir Dis; 132(5):1133-6 1985

Cigarette smoking is the major preventable cause of increased mortality and premature disability in the United States. Smoking is a proven cause of coronary heart disease, chronic bronchitis and emphysema, and lung and other cancers. Maternal smoking is associated with low infant birth weight, increased perinatal mortality, and several complications of pregnancy. Passive smoking is associated with increased incidence of lower respiratory tract infections in very young children and may increase the risk of lung cancer in the nonsmoker. Smoking cessation is difficult because of nicotine addiction and psychological and social factors. Physicians and other health professions must be active both in helping people quit smoking and in preventing nonsmokers from starting. We recommend a national effort focused on preventing young people from starting to smoke.

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171. PASSIVE SMOKING: A PUBLIC HEALTH PROBLEM (49 Refs)

Winters TH, DiFranza JR

Public Health Rev; 13(3-4):309-23 1985

Keywords (MeSH): Adult; Cardiovascular Diseases/etiology; Child; Environmental Exposure; Growth Disorders/etiology; Human; Lung/physiopathology; Lung Neoplasms/etiology; *Public Health; Respiratory Tract Diseases/etiology; Respiratory Tract Infections/etiology; Review; Risk; Tobacco Smoke Pollution/*adverse effects/prevention and control; United States.

172. SMOKING: HEALTH EFFECTS AND CONTROL (1) (87 Refs)

Fielding JE

University of California, Los Angeles, School of Public Health, CA 90024.

N Engl J Med; 313(8):491-8 1985

Keywords (MeSH): Adolescence; Adult; Arterial Occlusive Diseases/etiology; Birth Weight; Bladder Neoplasms/etiology; Body Weight; Cerebrovascular Disorders/etiology; Child; Coronary Disease/etiology; Esophageal Neoplasms/etiology; Female; Human; Infant, Newborn; Laryngeal Neoplasms/etiology; Lung Diseases, Obstructive/etiology; Lung Neoplasms/etiology; Male; Maternal-Fetal Exchange; Mouth Diseases/etiology; Mouth Neoplasms/etiology; Neoplasms/etiology; Pancreatic Neoplasms/etiology; Peptic Ulcer/etiology; Pregnancy; Review; *Smoking/prevention and control; Tobacco Smoke Pollution/adverse effects.

173. DOES BREATHING OTHER PEOPLE'S TOBACCO SMOKE CAUSE LUNG CANCER?

Wald NJ, Nanchahal K, Thompson SG, Cuckle HS

Department of Environmental and Preventive Medicine, St Bartholomew's Hospital Medical College, London.

Br Med J [Clin Res]; 293(6556):1217-22 1986

The available epidemiological studies of lung cancer and exposure to other people's tobacco smoke, in which exposure was assessed by whether or not a person classified as a non-smoker lived with a smoker, were identified and the results combined. There were 10 case-control studies and three prospective studies. Overall, there was a highly significant 35% increase in the risk of lung cancer among non-smokers living with smokers compared with non-smokers living with non-smokers (relative risk 1.35, 95% confidence interval 1.19 to 1.54). Part of this increase was almost certainly caused by the misclassification of some smokers as non-smokers. As smokers, who are more likely to get lung cancer than non-smokers, tend to live with smokers this misclassification probably exaggerated the estimated increase in risk. Adjustment for this error reduced the estimate to 30% (relative risk 1.30), but as people who live with non-smokers may still be exposed to other people's smoke this estimate was revised again to allow for the fact that a truly unexposed reference group was not used. The increase in risk among non-smokers living with smokers compared with a completely unexposed group was thus estimated as 53% (relative risk of 1.53). This analysis, and the fact that non-smokers breathe environmental tobacco smoke, which contains carcinogens, into their lungs and that the generally accepted view is that there is no safe threshold for the effect of carcinogens, leads to the conclusion that breathing other people's tobacco smoke is a cause of lung cancer. About a third of the cases of lung cancer in non-smokers who live with smokers, and about a quarter of the cases in non-smokers in general, may be attributed to such exposure.

174. CONTRIBUTION OF PASSIVE SMOKING TO RESPIRATORY CANCER

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Environ Health Perspect; 70:57-69 1986

This article reviews data from experimental and epidemiologic studies on passive smoking and makes 12 recommendations for further study. The physicochemical nature of passive smoke, the smoke inhaled by nonsmokers, differs significantly from the mainstream smoke inhaled by the active smoker. At present, measurement of urinary cotinine appears to be the best method of assessing exposures to passive smoking. Data indicate that the greater number of lung cancers in nonsmoking women is probably related to environmental tobacco smoke. Exposures in utero and very early in life to passive smoking may be important in relationship to the subsequent development of cancer and need further consideration. The short-term effects of environmental tobacco smoke on the cardiovascular system, especially among high-risk individuals, may be of greater concern than that of cancer and requires further study. Further study of increased risks of lung cancers in relation to environmental tobacco smoke exposure requires larger collaborative studies to identify lung cancer cases among nonsmokers, better delineation of pathology, and more careful selection of controls. In addition, studies of epithelial cells or specific cytology should be undertaken to determine evidence of cellular changes in relation to environmental tobacco smoke exposure. Animal inhalation studies with passive smoke should be initiated with respect to transplacental carcinogenesis, the relationship of sidestream smoke exposure with lung cancer, the induction of tumors in the respiratory tract and other organs, and the differences in the physicochemical natures of sidestream and mainstream smoke.

175. THE PASSIVE SMOKING-CANCER CONTROVERSY

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Banbury Rep; 23:343-60 1986

The epidemiology and biological plausibility of carcinogenesis resulting from passive smoking are reviewed, and unresolved questions related to the confounding effect of active smoking by 'nonsmokers,' problems in validating smoking status, the low-dose effect, and histologic types of lung cancer are discussed. At the present time it cannot be categorically affirmed or denied that passive smoking is a cause of cancer in humans. Evidence now available, however, is suggestive of a low-dose effect in the causation of cancers of the lung, nasal cavity, and brain. The main support for this position is the consistency of the findings in diverse populations in Greece, Japan, Germany, and the United States, and the biological plausibility provided by laboratory findings. Reports of the absence of association are fewer and have more serious shortcomings than reports of a positive association. The magnitude of the excess of cancer, if any, attributable to passive smoking is debatable. The discrepancy between reported relative risks and calculations based on equivalents of the number of cigarettes smoked actively may indicate bias in study designs, but may also call for the examination of alternative pathogenic mechanisms. The chapter concludes with a discussion by conference participants. (41 Refs)

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176. REVIEW OF EPIDEMIOLOGY IN RELATION TO PASSIVE SMOKING

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Arch Toxicol Suppl; 9:63-73 1986

Large segments of the population in many countries are exposed to environmental tobacco smoke which means that health effects resulting from this exposure are of great public health concern. Acute irritation of the eyes and airways as well as annoyance are probably the most common effects of passive smoking. Children with smoking parents run an increased risk of bronchitis and pneumonia. This seems to be related primarily to the smoking habits of the mother. Pulmonary function changes have also been reported in passive smokers but the data are not consistent. There are a few epidemiologic studies showing an increased risk of lung cancer in nonsmokers married to smokers. These studies may be affected with various types of biases and the association can not at present be regarded as causal. Particular attention should be put on the acquisition of precise and accurate exposure information in future epidemiologic investigations on health effects related to passive smoking. (79 Refs)

177. HEALTH RISKS OF PASSIVE SMOKING

Papier CM, Stellman SD
Department of Preventive and Social Medicine, Sackler School of Medicine, Tel Aviv University, Israel.
Women Health; 11(3-4):267-77 1986

Passive or involuntary smoking is the inhalation of smoke which escapes directly into the air from the lit end of a burning cigarette. This unfiltered smoke contains the same toxic components of the mainstream smoke inhaled directly by the smoker, including numerous carcinogens, many in greater concentrations. It has long been known that exposure to this type of smoke leads to increased respiratory and other adverse health conditions in non-smokers, especially children. During the past five years, evidence has been accumulating that risk of lung cancer is also higher, particularly in nonsmoking women whose husbands smoke. Despite uncertainties and differences in interpretation of various cancer studies, there is ample justification for public health measures now in place or proposed, such as restriction or elimination of smoking in the workplace and in public places.

178. PASSIVE SMOKING: A RISK FACTOR IN THE HOME ENVIRONMENT

Fidanza L, Franco G, Malamani T, Moscato G
Dipartimento di Medicina Preventiva, Universita degli Studi di Pavia.
G Ital Med Lav; 8(5-6):233-40 1986

Recent studies of the health effects of air pollution have pointed out the importance of environmental smoke as a pollution source in household. Several studies showed that children or adults living with parents or other adults who smoke have an increased risk of several effects: respiratory illness in children, changes of respiratory function in children and adults, risk of lung cancer and risk of other cancer than lung cancer. A few epidemiological surveys have studied the relationship between passive smoking and lung cancer. The available epidemiological studies (10 case-control studies, and 3 prospective studies) have been identified and the results combined. There was a 35% increase of risk of lung cancer among nonsmokers living with smokers as compared with nonsmokers living with nonsmokers. However, adjustments due to some degree of misclassification of some nonsmokers who may be still exposed to other people's smoke, increased the risk, which was thus estimated as 53%. Furthermore, cancer risk deriving from cumulative household exposures to cigarette smoke was evaluated. Overall cancer risk increased progressively with the number of household members who smoked. These results were observed for both smoking-related cancer and other sites cancer. Finally, although some of these results need to be confirmed, recent views believe that sufficient evidence is provided that breathing other people's tobacco smoke cause health impairment and lung cancer. (43 Refs)

179. OVERVIEW OF THE RISK OF RESPIRATORY CANCER FROM AIRBORNE CONTAMINANTS

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Environ Health Perspect; 70:9-15 1986

This overview on defining risk of respiratory cancer from airborne pollutants summarizes broad issues related to a number of the environmental agents that are discussed in the articles that follow. Lung cancer kills more than 100,000 people annually and is the major form of cancer in both sexes in middle age. Cigarette smoking is the major cause of respiratory cancer and must be taken into account in any study of the effect of an environmental agent on the risk of respiratory cancer, particularly at relatively low levels of excess risk (RR greater than 1.0 but less than 2.0). The agents considered in this series all have the potential for widespread community exposures, either because there is widespread long-term exposure (passive smoking), the agents are direct byproducts of energy consumption (organic particles), have ubiquitous production and use patterns (formaldehyde and fibers), or occur widely in natural settings (radon). Several issues - measurement of exposure, latency, confounding factors and bias, extrapolation from animals to humans, population at risk, and attributable risk - must be considered for each agent. A further issue related to exposure estimates is the relationship of exposure to actual dose. Understanding exposure some 25 to 40 years in the past is important because of the prolonged latency period in the development of respiratory cancers. To the degree that these agents act synergistically with smoking, the reduction of smoking or of exposure to these agents may have greater public health consequences than would be anticipated from the directly measured attributable risk of each of these agents separately.

180. SMOKING AND OCCUPATIONAL LUNG CANCER

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Cancer Prevention. Strategies in the Workplace. Becker CE, Coye MJ, eds. Washington, DC, Hemisphere Publishing Corporation, p. 121-42, 1986.

The importance of smoking as a contributor to occupational lung disease has been recognized in the past 15 yr. Several studies indicate a synergistic or multiplicative effect of smoking and occupational toxin exposures. Because certain cancers would not have occurred without the combination of occupational exposure and cigarette smoking, these cancers must be considered to be occupation-related cancers. Since industry and workman's compensation are financially responsible for occupationally-induced illnesses, smoking becomes a concern of the employer. Recent studies indicate that passive smoke exposure may also result in the impairment of pulmonary function and, possibly, lung cancer in the nonsmoker. A workplace in which the nonsmoker is exposed to cigarette smoke is, therefore, considered hazardous. A few industries have cigarette smoking control policies. With respect to this, smoking and occupational lung cancer are discussed under the following headings: smoking and cancer; how does ciga-

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rette smoking cause cancer; smoking in the workplace; smoking and workplace cancer (asbestos; uranium ore and metal miners; arsenic (copper smelter) workers; chloromethyl ethers: does smoking protect against occupational cancer; lung cancer in workers: occupation vs smoking; factors possibly confounding the relationship between smoking, occupation and cancer); passive smoking and lung cancer (mainstream vs sidestream cigarette smoke; pathology and pathogenesis of lung cancer in smokers vs nonsmokers; evidence that nonsmokers inhale cigarette smoke; general hazards of passive smoking; lung cancer and passive smoke exposure; passive smoking and workplace cancer); and strategies to control workplace smoking (smoking cessation strategies; control of passive smoke exposure). Smoking in the workplace can be a cause of occupational cancer in smokers exposed to other occupational chemicals and probably in nonsmokers exposed to tobacco smoke of other workers. Simultaneous control of workplace smoking and exposure to occupational toxins is necessary to minimize the likelihood of occupational cancer. Control of workplace smoking should be a combined effort of management and labor, and would be greatly benefited by support from OSHA. (78 Refs)

181. CIGARETTE SMOKING AND LUNG DISEASE

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Am J Med Sci; 291(2):130-42 1986

Despite major changes in the patterns of cigarette consumption in the United States in the last two decades, cigarette smoking remains a widespread practice. Many studies show a strong association between cigarette smoking and chronic obstructive lung disease, but only recently have mechanisms been elucidated to explain this association, particularly in the case of emphysema. The exact mechanism of chronic bronchitis is less well defined. The mortality rate for lung cancer continues to increase in this country, particularly among women. Compelling evidence associates lung cancer with cigarette smoking, although animal models have not been very successful. Recently, concern has been raised about the effects of passive smoking. There are substantial data available to suggest that passive smoking results in pulmonary infections and abnormal pulmonary function in children of smokers. The data are less clear-cut for pulmonary functional impairment in adults and for lung cancer in the spouses of smokers. Although the concept of 'safer' cigarettes has been widely accepted by the American public, these cigarettes may not be as safe as is widely assumed. Clearly, they are far inferior to total smoking cessation. Nevertheless, smoking cessation remains difficult for many Americans, despite a number of methods that have been used to help smokers quit. Nicotine chewing gum, which has recently become available for use in the United States, has shown some efficacy in helping well-motivated, nicotine-dependent smokers quit smoking. Although some inroads have been made into the occurrence of smoking related diseases in the United States, many unnecessary deaths continue to occur. (81 Refs)

182. 1986 SURGEON GENERAL'S REPORT: THE HEALTH CONSEQUENCES OF INVOLUNTARY SMOKING

Anonymous

MMWR; 35(50):769-70 1986

Keywords (MeSH): Human; Lung Neoplasms/etiology; Respiratory Tract Diseases/etiology; Tobacco Smoke Pollution/*adverse effects/prevention and control; United States.

183. ENVIRONMENTAL TOBACCO SMOKE: MEASURING EXPOSURES AND ASSESSING HEALTH EFFECTS

Committee on Passive Smoking

Board on Environmental Studies and Toxicology, National Res. Council, Washington, DC

Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. Washington, DC. National Academy Press. 367 p., 1986.

This report, by the Committee on Passive Smoking of the NRC Commission on Life Sciences, deals with present data on the health effects of environmental tobacco smoke (ETS). These topics are discussed: types and composition of ETS; possible measures of exposure; in vitro and in vivo assays of mutagenicity; acute, noxious effects; lung function studies; lung cancer incidence; other cancers; cardiovascular disease; and developmental effects in utero and in children. In addition to presenting the available data, problems and inadequacies in the area are discussed and suggestions are made for additional studies. Since there are more than 3000 components of tobacco smoke, measures of exposure are difficult; there are major differences between mainstream and side stream smoke, and the human pharmacokinetics of these components vary widely. Many measures are presented, including nicotine/cotinine and urinary mutagens, but none are clearly ideal. Extrapolation from smokers to passive smokers may not always be appropriate because of the differences in type of smoke inhaled and innate metabolism. Most of the health effects are derived from chronic studies of individuals living for long periods with spouses and other family members who smoke; relatively little is known of the effects of intermittent or workplace exposure. Among nonsmoking spouses, an increase in the incidence of lung cancer of about 30% has been seen in a number of studies. Children of smokers exhibit a 20-80% increase in the incidence of wheezes, coughing, and sputum production. Finally, there is evidence of reduced growth rates for children of smokers in terms of lung function. Other parameters of health have not shown consistent evidence of effects. Areas pointed out for research emphasis include in utero effects, other forms of cancer, cardiovascular studies, and the whole area of quantification of exposure.

184. PASSIVE SMOKING AND LUNG CANCER (53 Refs)

Blot WJ, Fraumeni JF Jr

Epidemiology and Biostatistics Program, National Cancer Institute, Bethesda, MD 20892.

J Natl Cancer Inst; 77(5):993-1000 1986

Keywords (MeSH): Environmental Exposure; Great Britain; Hong Kong; Human; Japan; Lung Neoplasms/*etiology/mortality; Review; Risk; Sweden; *Tobacco Smoke Pollution; United States.

185. PASSIVE SMOKING AND LUNG CANCER

Saracci R

International Agency for Research on Cancer, Lyon, France.

IARC Sci Publ; (74):173-82 1986

Keywords (MeSH): Epidemiologic Methods; Female; Human; Lung Neoplasms/*etiology/EP; Male; Tobacco Smoke Pollution/*adverse effects.

186. NHMRC REPORT. EFFECTS OF PASSIVE SMOKING ON HEALTH

Anonymous

Med J Aust; 145(8):404-5 1986

Keywords (MeSH): Cardiovascular Diseases/etiology; Child Welfare; *Health; Human; Lung Neoplasms/etiology; Respiratory Tract Diseases/etiology; Risk; Tobacco Smoke Pollution/*adverse effects.

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187. AMERICAN ACADEMY OF PEDIATRICS. INVOLUNTARY SMOKING - A HAZARD TO CHILDREN. COMMITTEE ON ENVIRONMENTAL HAZARDS
Anonymous
Pediatrics; 77(5):755-7 1986

Keywords (MeSH): Child; Coronary Disease/etiology; Human; Infant; Lung Neoplasms/etiology; Pediatrics; Respiratory Tract Infections/*etiology; Societies, Medical; Tobacco Smoke Pollution/*adverse effects/analysis; United States.

188. TOBACCO: AN OVERVIEW OF HEALTH EFFECTS (33 Refs)
Doll R
University of Oxford, U.K.
IARC Sci Publ, 74:11-22 1986

Keywords (MeSH): Cardiovascular Diseases/etiology; Human; Neoplasms/etiology; Review; Risk; *Smoking; Tobacco Smoke Pollution.

189. PASSIVE SMOKING AND LUNG CANCER: EPIDEMIOLOGICAL EVIDENCE AND ONGOING INTERNATIONAL COLLABORATIVE STUDIES
Riboli E
International Agency for Research on Cancer, Lyon, France.
Toxicol Lett; 35(1):19-27 1987

Epidemiological studies which investigated the association between lung cancer in non-smokers and exposure to tobacco smoke are reviewed. Discordant results are discussed in the light of biases and misclassification. Design, materials and methods are presented of an ongoing international collaborative study to explore the relationship between recent exposure to passive smoking (P.S.), as assessed by interview, and cotinine and thiocyanate levels in urine.

190. PASSIVELY INHALED TOBACCO SMOKE: A CHALLENGE TO TOXICOLOGY AND PREVENTIVE MEDICINE
Remmer H
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Arch Toxicol; 61(2):89-104 1987

The difficulties in defining the exposure of a passive smoker might explain the controversial results regarding an association between passive smoking on one hand and lung cancer, tumors of all sites and ischemic heart diseases on the other. The plausibility of these epidemiological observations will be discussed in the light of analytical, toxicological, biochemical and oncological data. The minute amounts of nicotine and particulate matter, even the much higher concentrations of volatile substances, such as nitrosamines, NOx, acroleine and formaldehyde, present in diluted sidestream compared to mainstream smoke and inhaled by involuntarily smoking people, cannot explain their relatively high cancer risk. It is plausible if one considers the high capacity of cigarette smoke to induce drug metabolizing enzymes. Diluted sidestream smoke, however, lacks compounds which induce several iso-enzymes of cyt. P-450 monooxygenase in the tissues. The best evidence is the up to 100-fold increase in placental enzymes if pregnant women smoke, whereas passively inhaled tobacco smoke is ineffective as inducer. The small amounts of paternal smoke inhaled by pregnant women, containing teratogenic and carcinogenic compounds, which are supposedly not detoxified in the placenta, seem to explain the higher risk for malformations of the fetus and the same or even increased risk for perinatal mortality, compared with the outcome of pregnancy if the mother smoked. The induction of placental enzymes very probably protects the fetus against the much higher amounts of toxic agents inhaled by the smoking mother. The increased activity of placental enzymes seems to be a model for the probably greater capacity of certain cyt. P-450 iso-enzymes in the lung and other tissues to convert carcinogens to inactive metabolites when the individual smokes actively. It is well known that concomitant administration of carcinogens with inducing agents inhibits tumor growth in animals because of a shift in the metabolism which favours the formation of ineffective substances. The negligible amounts of nicotine and CO in passively inhaled tobacco smoke cannot be responsible for the surprisingly high risk for ischemic heart diseases of passive smokers. A plausible explanation is offered by experiments with doves and chicken, which develop atherosclerotic lesions due to the action of carcinogens which are metabolized by certain inducible cyt. P-450 iso-enzymes in the aortic wall. Much circumstantial evidence will be presented, indicating that PAHs, contrary to the propagated opinion, play a minor role for the initiation of cancer in active smokers.(ABSTRACT TRUNCATED AT 400 WORDS) (186 Refs)

191. IS PASSIVE SMOKING INCREASING CANCER RISK?
Vainio H
International Agency for Research on Cancer, Lyon, France.
Scand J Work Environ Health; 13(3):193-6 1987

Although the amounts of environmental tobacco smoke absorbed by passive smokers are small in comparison to those absorbed by active smokers, the fact that (almost) everybody in modern society is exposed makes it an important public health problem. The many published epidemiologic studies are all consistent with a 30% increase in the risk of lung cancer. This increase is plausible in relation to the exposure levels derived from various biological dose indicators. The risks of passive smokers are, of course, smaller than those of active smokers, but it is generally accepted that involuntary risks should be much smaller than those that are self-inflicted. Even a relative risk for lung cancer of 1.3 due to passive smoking would constitute an increase on the order of 1 in 1,000 in the lifetime risk. Normally, this level would be considered 'unacceptable,' and preventive measures should be taken. (36 Refs)

192. EPIDEMIOLOGICAL STUDIES OF PASSIVE SMOKING (71 Refs)
Preston-Martin S, Dodet B, Saracci R
University of Southern California School of Medicine, Department of Preventive Medicine, Los Angeles 90033.
IARC Sci Publ; (81):69-84 1987

Keywords (MeSH): Air Pollutants, Environmental/*poisoning; Epidemiologic Methods; Human; Lung Diseases/*chemically induced; Lung Neoplasms/*chemically induced; Review; Review, Tutorial; Tobacco Smoke Pollution/*adverse effects.

193. [RADIOACTIVITY OF CIGARETTE SMOKE AND LUNG CANCER] (21 Refs)
Fu H, Wu KG, Cobb JC
Chung Hua Yu Fang I Hsueh Tsa Chih; 21(3):152-4 1987

Keywords (MeSH): Human; Lung Neoplasms/*chemically induced; Polonium/*analysis/poisoning; Review Literature; Review; Tobacco Smoke Pollution/adverse effects/*analysis.

194. TOBACCO SMOKE: THE ENVIRONMENTAL AIR POLLUTANT MOST DANGEROUS TO MAN (30 Refs)
Remmer H
Institut fur Toxikologie der Universitat Tubingen.
Dtsch Med Wochenschr; 112(26):1054-9 1987

Keywords (MeSH): Cocarcinogenesis; Female; Human; Hydroxylases/biosynthesis; Liver/enzymology; Lung Neo-

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